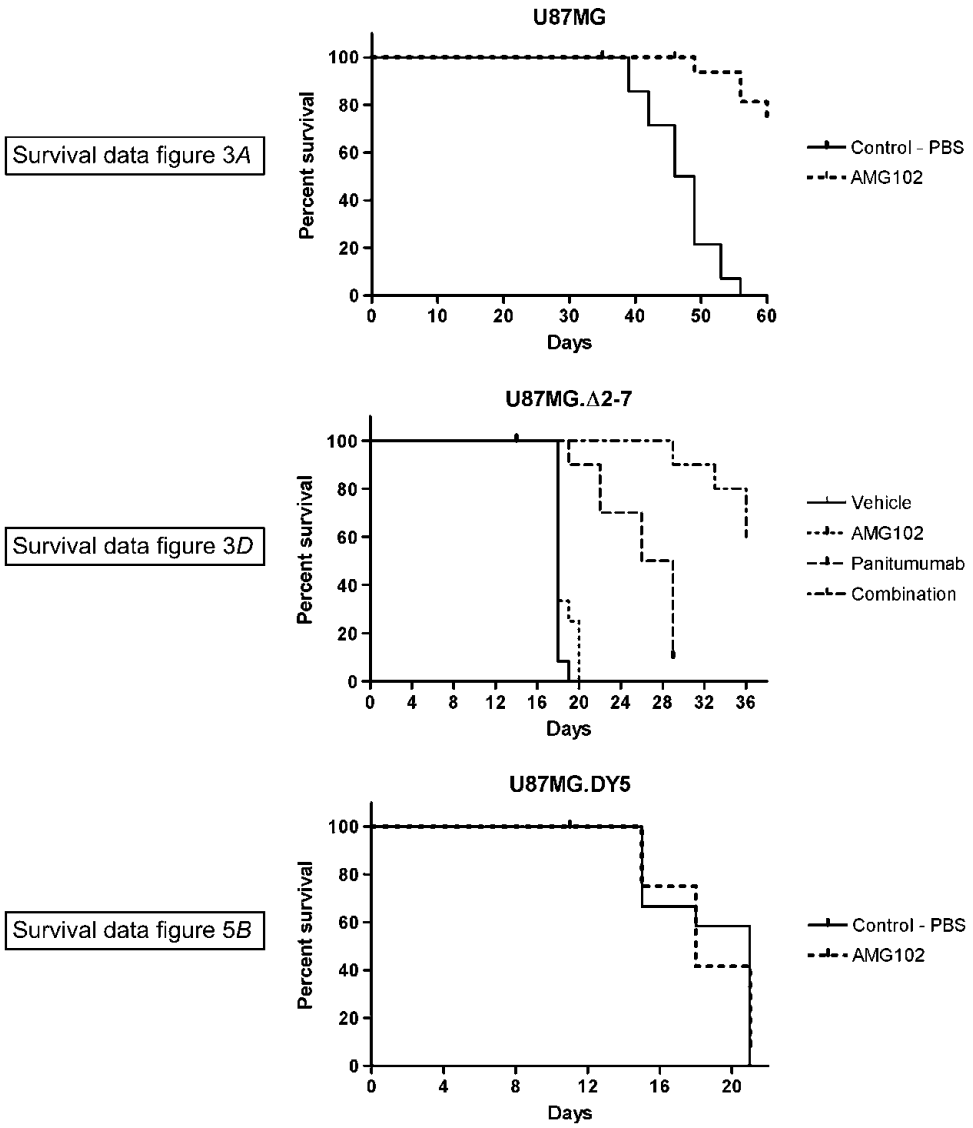

Figure W1. Survival data from xenograft studies. Survival analyses for the therapy studies described in Figures 3, *A* and *D*, and 5*B*. Survival was evaluated using the technique of Kaplan-Meier with comparisons between groups analyzed by log-rank test. A combined end point of moribund condition or tumor volume reaching 1000 mm^3 in mice treated with vehicle.

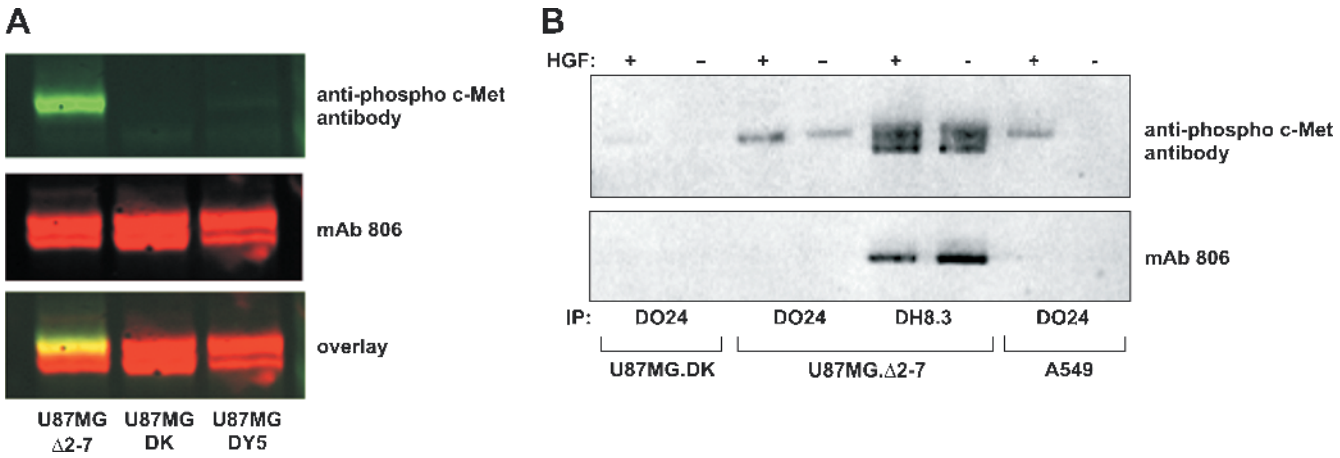

Figure W2. Cross-reactivity between phospho–c-Met antibodies and constitutively active de2-7 EGFR. (A) U87MG.Δ2-7, U87MG.DK, and U87MG.DY5 whole-cell lysates were Western blotted with mAb 806 (anti-EGFR; middle panel) or anti–phospho c-Met (upper panel; rabbit (polyclonal) phosphospecific anti–c-Met [pYpYpY$^{1230/1234/1235}$] from Invitrogen). De2-7 EGFR–related proteins can be seen in all cell lines. In contrast, a p–c-Met band can only be seen in U87MG.Δ2-7. Because the de2-7 EGFR is not phosphorylated in U87MG.DK and U87MG.DY5 cells and because p–c-Met and de2-7 EGFR comigrate (lower panel), it is impossible to determine whether the band seen in the upper panel is p–c-Met or the antibody cross-reacting with de2-7 EGFR. (B) U87MG.Δ2-7 and U87MG.DK cells were immunoprecipitated with DO24 (c-Met–specific) or DH8.3 (de2-7 EGFR–specific) and Western blotted with phospho–c-Met (upper panel) or mAb 806 (lower panel). A549 cells ± HGF were used as a control. As can be clearly seen, the de2-7 EGFR immunoprecipitated by DH8.3 cross-reacted with the anti–p–c-Met antibody. Specific phosphorylation of c-Met could only be observed after immunoprecipitation with DO24. Similar results were obtained using three different phosphospecific c-Met antibodies. Thus, these antibodies cannot be used to determine levels of phosphorylated c-Met by immunohistochemistry in tissues expressing the de2-7 EGFR.